CHAPTER

4

Neural Conduction and Synaptic Transmission

How Neurons Send and Receive Signals

Visual BRAIN

Explore the **Neural Conduction and Synaptic Transmission module** in MyPsychLab.

4.1 Resting Membrane Potential
4.2 Generation and Conduction of Postsynaptic Potentials
4.3 Integration of Postsynaptic Potentials and Generation of Action Potentials
4.4 Conduction of Action Potentials
4.5 Synaptic Transmission: Chemical Transmission of Signals among Neurons
4.6 Neurotransmitters
4.7 Pharmacology of Synaptic Transmission and Behavior

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LEARNING OBJECTIVES

- **LO1** Describe the resting membrane potential and its ionic basis.
- **LO2** Explain postsynaptic potentials.
- **LO3** Describe the summation of postsynaptic potentials.
- **LO4** Explain how an action potential is normally triggered.
- **LO5** Describe how action potentials are conducted along axons.
- **LO6** Discuss the structure and variety of synapses.
- **LO7** Name and explain the classes of neurotransmitters.
- **LO8** Name and compare different neurotransmitters.
- **LO9** Discuss 3 examples of how drugs have been used to influence neurotransmission.

Chapter 3 introduced you to the anatomy of neurons. This chapter introduces you to their function—how neurons conduct and transmit electrochemical signals through your nervous system. It begins with a description of how signals are generated in resting neurons; then, it follows the signals as they are conducted through neurons and transmitted across synapses to other neurons. It concludes with a discussion of how drugs are used to study the relation between synaptic transmission and behavior. The following case study of a patient with Parkinson's disease will help you appreciate why a knowledge of neural conduction and synaptic transmission is an integral part of biopsychology (Klawans, 1990).

*The Lizard, a Case of Parkinson's Disease

"I have become a lizard," he began. "A great lizard frozen in a dark, cold, strange world."

His name was Roberto Garcia d'Orta. He was a tall thin man in his sixties, but like most patients with Parkinson's disease, he appeared to be much older than his actual age. Not many years before, he had been an active, vigorous business man. Then it happened—not all at once, not suddenly, but slowly, subtly, insidiously. Now he turned like a piece of granite, walked in slow shuffling steps, and spoke in a monotonous whisper.

What had been his first symptom?

A tremor.

Had his tremor been disabling?

"No," he said. "My hands shake worse when they are doing nothing at all"—a symptom called *tremor-at-rest.*

The other symptoms of Parkinson's disease are not quite so benign. They can change a vigorous man into a lizard. These include rigid muscles, a marked poverty of spontaneous movements, difficulty in starting to move, and slowness in executing voluntary movements once they have been initiated.

The term "reptilian stare" is often used to describe the characteristic lack of blinking and the widely opened eyes gazing out of a motionless face, a set of features that seems more reptilian than human. Truly a lizard in the eyes of the world.

What was happening in Mr. d'Orta's brain? A small group of nerve cells called the *substantia nigra* (black substance) were unaccountably dying. These neurons make a particular chemical called dopamine, which they deliver to another part of the brain, known as the *striatum.* As the cells of the substantia nigra die, the amount of dopamine they can deliver goes down. The striatum helps control movement, and to do that normally, it needs dopamine.

Although dopamine levels are low in Parkinson's disease, dopamine is not an effective treatment because it does not readily penetrate the blood–brain barrier. However, knowledge of dopaminergic transmission has led to the development of an effective treatment: *L-dopa,* the chemical precursor of dopamine, which readily penetrates the blood–brain barrier and is converted to dopamine once inside the brain.

Mr. d'Orta's neurologist prescribed L-dopa, and it worked. He still had a bit of tremor; but his voice became stronger, his feet no longer shuffled, his reptilian stare faded away, and he was once again able to perform with ease many of the activities of daily life (e.g., eating, bathing, writing, speaking, and even making love with

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his wife). Mr. d'Orta had been destined to spend the rest of his life trapped inside a body that was becoming increasingly difficult to control, but his life sentence was repealed—at least temporarily.

Mr. d'Orta's story does not end here. You will learn what ultimately happened to him in Chapter 10. Meanwhile, keep him in mind while you read this chapter: His case illustrates why knowledge of the fundamentals of neural conduction and synaptic transmission is a must for any biopsychologist.

4.1 Resting Membrane Potential

As you are about to learn, the key to understanding how neurons work—and how they malfunction—is the membrane potential. The **membrane potential** is the difference in electrical charge between the inside and the outside of a cell.

RECORDING THE MEMBRANE POTENTIAL

To record a neuron's membrane potential, it is necessary to position the tip of one electrode inside the neuron and the tip of another electrode outside the neuron in the extracellular fluid. Although the size of the extracellular electrode is not critical, it is paramount that the tip of the intracellular electrode be fine enough to pierce the neural membrane without severely damaging it. The intracellular electrodes are called **microelectrodes**; their tips are less than one-thousandth of a millimeter in diameter—much too small to be seen by the naked eye.

When both electrode tips are in the extracellular fluid, the voltage difference between them is zero. However, when the tip of the intracellular electrode is inserted into a neuron, a steady potential of about −70 millivolts (mV) is recorded. This indicates that the potential inside the resting neuron is about 70 mV less than that outside the neuron. This steady membrane potential of about −70 mV is called the neuron's **resting potential.** In its resting state, with the −70 mV charge built up across its membrane, a neuron is said to be *polarized.*

IONIC BASIS OF THE RESTING POTENTIAL

Like all salts in solution, the salts in neural tissue separate into positively and negatively charged particles called **ions.** There are many different kinds of ions in neurons, but this discussion focuses on only two of them: sodium ions and potassium ions. The abbreviations for sodium ions (Na^+) and potassium ions (K^+) are derived from their latin names: natrium (Na) and kalium (K). The plus signs indicate that each Na^+ and K^+ ion carries a single positive charge.

In resting neurons, there are more Na^+ ions outside the cell than inside, and more K^+ ions inside than outside. These unequal distributions of Na^+ and K^+ ions are maintained even though there are specialized pores, called **ion channels,** in neural membranes through which ions can pass. Each type of ion channel is specialized for the passage of particular ions (e.g., Na^+ or K^+).

There is substantial pressure on Na^+ ions to enter the resting neurons. This pressure is of two types. First, is the *electrostatic pressure* from the resting membrane potential: Because opposite charges attract, the −70mV charge attracts the positively charged Na^+ ions into resting neurons. Second is the pressure from *random motion* for Na^+ ions to move down their *concentration gradient.* Let me explain. Like all ions in solution, the ions in neural tissue are in constant random motion, and particles in random motion tend to become evenly distributed because they are more likely to move down their *concentration gradients* than up them; that is, they are more likely to move from areas of high concentration to areas of low concentration than vice versa.

So, why then do Na^+ ions under electrostatic pressure and pressure from random movement not come rushing into neurons, thus reducing the resting membrane potential? The answer is simple: The sodium ion channels in resting neurons are closed, thus greatly reducing the flow of Na^+ ions into the neuron. In contrast, the potassium channels are open in resting neurons, but only a few K^+ ions exit because they are largely held inside by the negative resting membrane potential.

In the 1950s, Alan Hodgkin and Andrew Huxley became interested in the stability of the resting membrane potential. Some Na^+ ions do manage to enter resting neurons despite the closed sodium channels and some K^+ ions do exit; then why does the resting membrane potential stay fixed? In a series of clever experiments, for which they were awarded Nobel Prizes, Hodgkin and Huxley discovered the answer. At the same rate that Na^+ ions leaked into resting neurons, other Na^+ ions were actively transported out; and at the same rate that K^+ ions leaked out of resting neurons, other K^+ ions were actively transported in. Such ion transport is performed by mechanisms in the cell membrane that continually exchange three Na^+ ions inside the neuron for two K^+ ions outside. These transporters are commonly referred to as **sodium–potassium pumps.**

Since the discovery of sodium–potassium pumps, several other classes of **transporters** (mechanisms in the membrane of a cell that actively transport ions or molecules across the membrane) have been discovered (e.g., Tzingounis & Wadiche, 2007). You will encounter more of them later in this chapter.

Figure 4.1 summarizes the status of Na^+ and K^+ ions in resting neurons. Now that you understand the basic properties of resting neurons, you are prepared to consider how neurons respond to input.

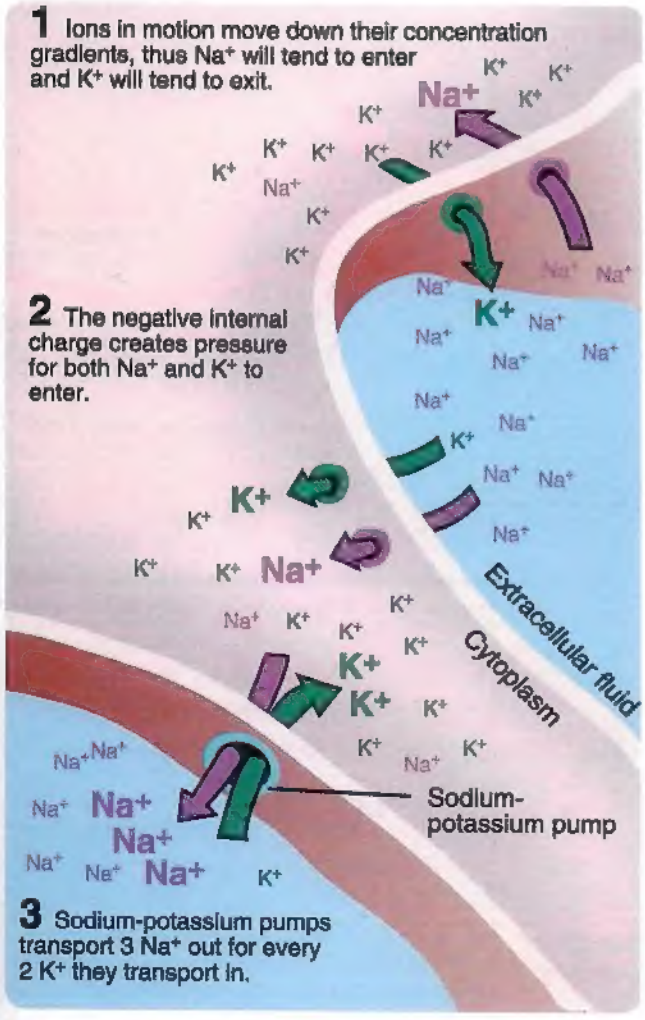

FIGURE 4.1 Three factors that influence the distribution of Na^+ and K^+ ions across the neural membrane.

4.2 Generation and Conduction of Postsynaptic Potentials

When neurons fire, they release from their terminal buttons chemicals called *neurotransmitters*, which diffuse across the synaptic clefts and interact with specialized receptor molecules on the receptive membranes of the next neurons in the circuit. When neurotransmitter molecules bind to postsynaptic receptors, they typically have one of two effects, depending on the neurotransmitter, receptor, and postsynaptic neuron in question. They may **depolarize** the receptive membrane (decrease the resting membrane potential, from −70 to −67 mV, for example) or they may **hyperpolarize** it (increase the resting membrane potential, from −70 to −72 mV, for example). The ionic mechanisms mediating postsynaptic potentials are different in different kinds of neurons so I will not discuss them here.

Postsynaptic depolarizations are called **excitatory postsynaptic potentials (EPSPs)** because, as you will soon learn, they increase the likelihood that the neuron will fire. Postsynaptic hyperpolarizations are called **inhibitory postsynaptic potentials (IPSPs)** because they decrease the likelihood that the neuron will fire. Both EPSPs and IPSPs are **graded responses.** This means that the amplitudes of EPSPs and IPSPs are proportional to the intensity of the signals that elicit them: Weak signals elicit small postsynaptic potentials, and strong signals elicit large ones.

EPSPs and IPSPs travel passively from their sites of generation at synapses, usually on the dendrites or cell body, in much the same way that electrical signals travel through a cable. Accordingly, the transmission of postsynaptic potentials has two important characteristics. First, it is rapid—so rapid that it can be assumed to be instantaneous for most purposes. It is important not to confuse the duration of EPSPs and IPSPs with their rate of transmission; although the duration of EPSPs and IPSPs varies considerably, all postsynaptic potentials, whether brief or enduring, are transmitted at great speed. Second, the transmission of EPSPs and IPSPs is *decremental*: EPSPs and IPSPs decrease in amplitude as they travel through the neuron, just as a sound wave loses amplitude (the sound grows fainter) as it travels through air. Most EPSPs and IPSPs do not travel more than a couple of millimeters from their site of generation before they fade out; thus, few travel very far along an axon.

4.3 Integration of Postsynaptic Potentials and Generation of Action Potentials

The postsynaptic potentials created at a single synapse typically have little effect on the firing of the postsynaptic neuron (Bruno & Sakmann, 2006). The receptive areas of most neurons are covered with thousands of synapses, and whether or not a neuron fires is determined by the net effect of their activity. More specifically, whether or not a neuron fires depends on the balance between the excitatory and inhibitory signals reaching its axon. It was once believed that action potentials were generated at the **axon hillock** (the conical structure at the junction between the cell body and the axon), but they are actually generated in the adjacent section of the axon, called the **axon initial segment** (Bender & Trussell, 2012; Grubb & Burronne, 2010; Kole & Stuart, 2012).

The graded EPSPs and IPSPs created by the action of neurotransmitters at particular receptive sites on a neuron's membrane are conducted instantly and decrementally to the axon initial segment. If the sum of the depolarizations and hyperpolarizations reaching the axon initial segment at any time is sufficient to depolarize the membrane to a level referred to as its **threshold of excitation**—usually about −65 mV—an action potential

is generated. The **action potential (AP)** is a massive but momentary—lasting for 1 millisecond—reversal of the membrane potential from about −70 to about +50 mV. Unlike postsynaptic potentials, action potentials are not graded responses; their magnitude is not related in any way to the intensity of the stimuli that elicit them. To the contrary, they are **all-or-none responses**; that is, they either occur to their full extent or do not occur at all. See Figure 4.2 for an illustration of an EPSP, an IPSP, and an AP. Although many neurons display APs of the type illustrated in Figure 4.2, others do not—for example, some neurons display APs that are longer, that have lower amplitude, or that involve multiple spikes.

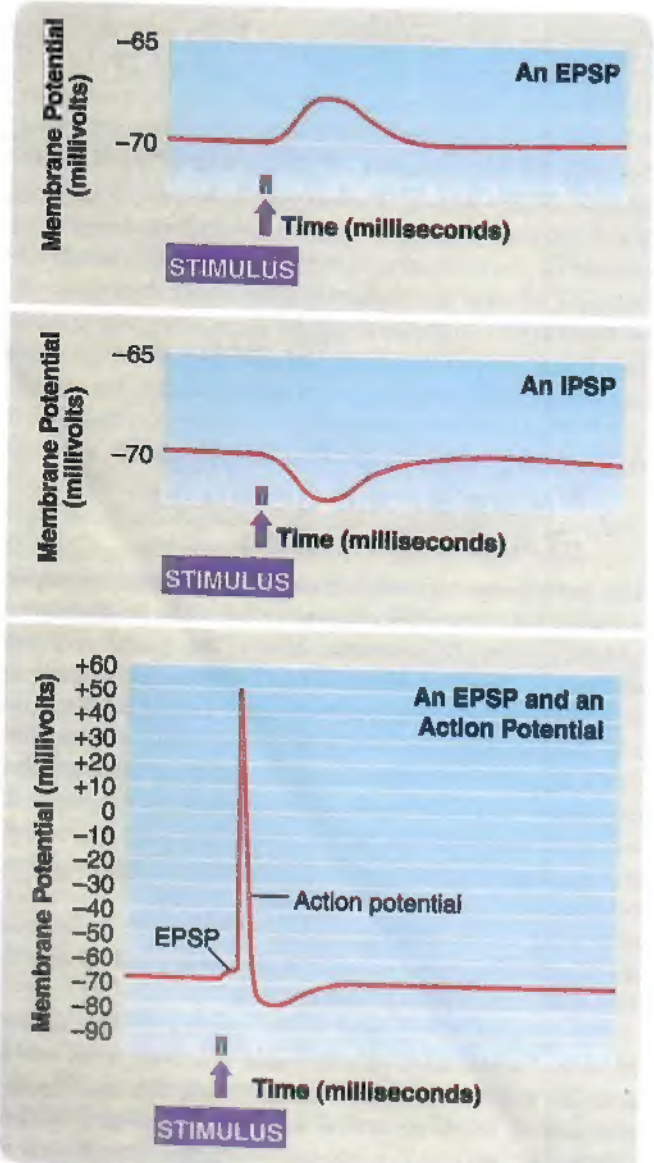

FIGURE 4.2 An EPSP, an IPSP, and an EPSP followed by a typical AP.

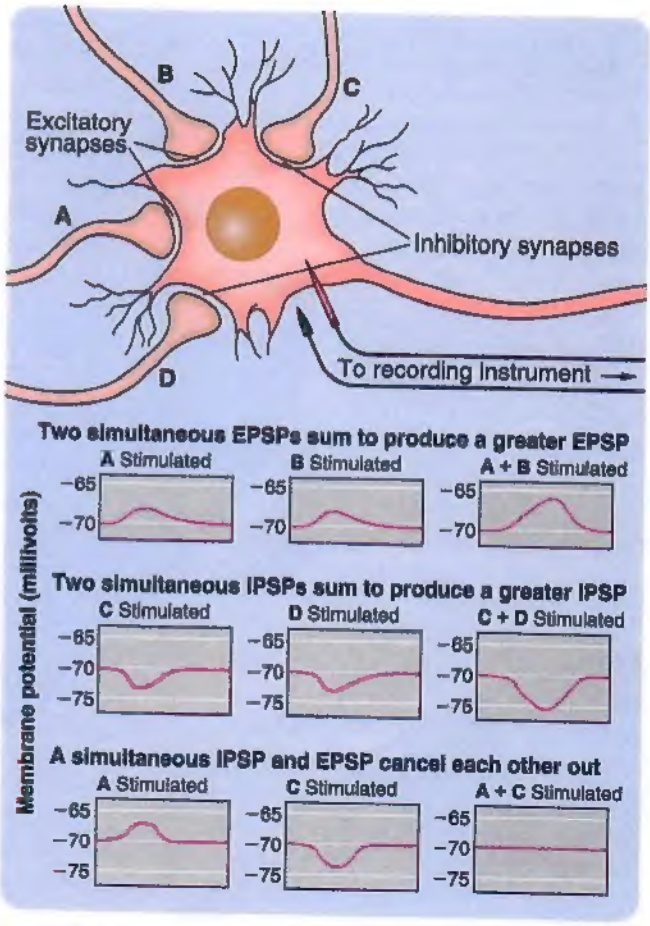

FIGURE 4.3 The three possible combinations of spatial summation.

In effect, each multipolar neuron adds together all the graded excitatory and inhibitory postsynaptic potentials reaching its axon and decides to fire or not to fire on the basis of their sum. Adding or combining a number of individual signals into one overall signal is called **integration.** Neurons integrate incoming signals in two ways: over space and over time.

Figure 4.3 shows the three possible combinations of **spatial summation.** It shows how local EPSPs that are produced simultaneously on different parts of the receptive membrane sum to form a greater EPSP, how simultaneous IPSPs sum to form a greater IPSP, and how simultaneous EPSPs and IPSPs sum to cancel each other out.

Figure 4.4 illustrates **temporal summation.** It shows how postsynaptic potentials produced in rapid succession at the same synapse sum to form a greater signal. The reason that stimulations of a neuron can add together over time is that the postsynaptic potentials they produce often outlast them. Thus, if a particular synapse is activated and then activated again before the original postsynaptic

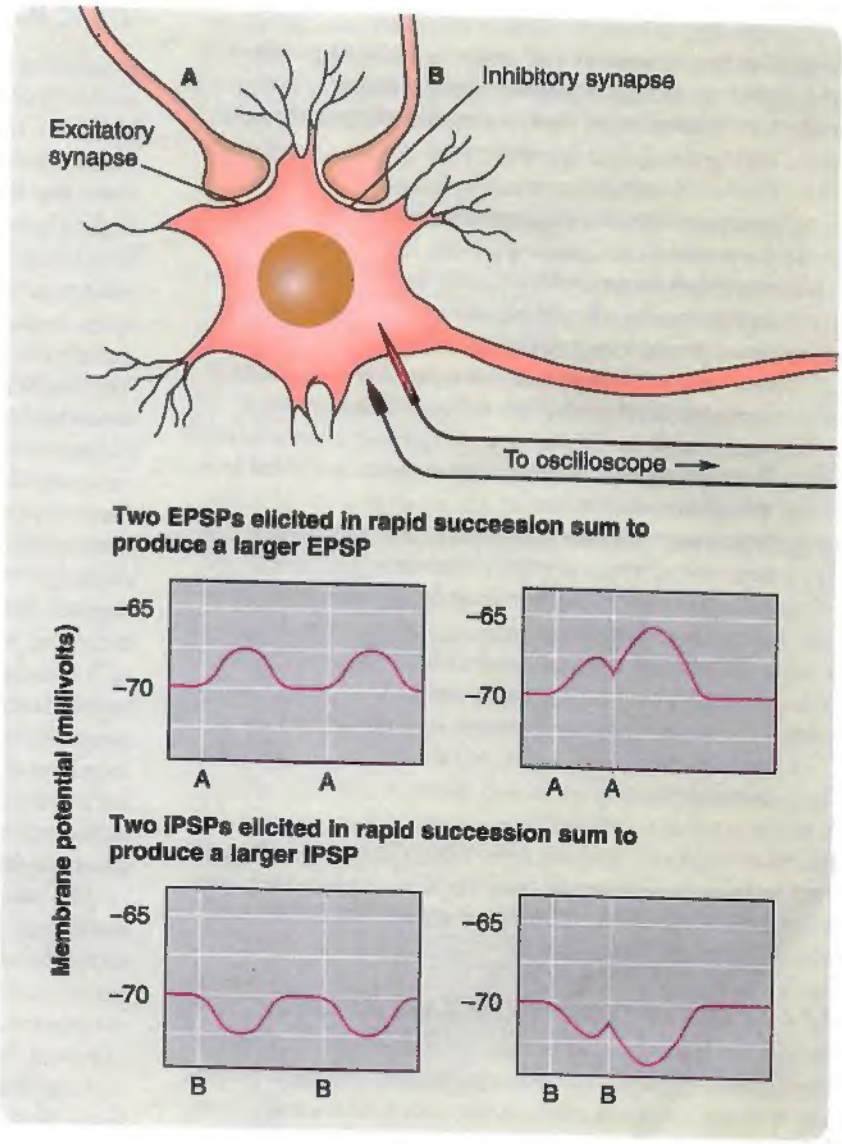

FIGURE 4.4 The two possible combinations of temporal summation.

potential has completely dissipated, the effect of the second stimulus will be superimposed on the lingering postsynaptic potential produced by the first. Accordingly, it is possible for a brief subthreshold excitatory stimulus to fire a neuron if it is administered twice in rapid succession. In the same way, an inhibitory synapse activated twice in rapid succession can produce a greater IPSP than that produced by a single stimulation.

Each neuron continuously integrates signals over both time and space as it is continually bombarded with stimuli through the thousands of synapses covering its dendrites and cell body. Although schematic diagrams of neural circuitry rarely show neurons with more than a few representative synaptic contacts, most neurons receive thousands of such contacts.

The location of a synapse on a neuron's receptive membrane has long been assumed to be an important factor in determining its potential to influence the neuron's firing. Because EPSPs and IPSPs are transmitted decrementally, synapses near the axon trigger zone have been assumed to have the most influence on the firing of the neuron. However, it has been demonstrated that some neurons have a mechanism for amplifying dendritic signals that originate far from their axon initial segments (Branco, 2011).

In some ways, the firing of a neuron is like the firing of a gun. Both reactions are triggered by graded responses. As a trigger is squeezed, it gradually moves back until it causes the gun to fire; as a neuron is stimulated, it becomes less polarized until the threshold of excitation is reached and firing occurs. Furthermore, the firing of a gun and neural firing are both all-or-none events. Just as squeezing a trigger harder does not make the bullet travel faster or farther, stimulating a neuron more intensely does not increase the speed or amplitude of the resulting action potential.

Thinking Creatively

Simulate *Virtual Brain: Neural Conduction* at **MyPsychLab**

SCAN YOUR BRAIN

Before you learn how action potentials are conducted along the axon, pause here to make sure you understand how action potentials are created. Fill in each blank with the most appropriate term. The correct answers are provided at the end of the exercise. Before proceeding, review material related to your errors and omissions.

1. Roberto Garcia d'Orta referred to himself as "a great lizard frozen in a dark, cold, strange world." He suffered from _____.
2. Tremor-at-rest is a symptom of _____.
3. Microelectrodes are required to record a neuron's resting _____.
4. The _____ is about –70 mV.

5. In its resting state, a neuron is said to be _____.
6. Two factors pressure Na^+ ions to enter resting neurons: random _____ and electrostatic pressure.
7. In the resting state, there is a greater concentration of Na^+ ions _____ the neuron than _____ the neuron.
8. *Natrium* is Latin for _____.
9. Ions pass through neural membranes via specialized pores called _____.
10. From their studies, Hodgkin and Huxley inferred the existence of _____ in neural membranes, the first neural transporters to be discovered.
11. Neurotransmitters typically have one of two effects on postsynaptic neurons: They either depolarize them or _____ them.
12. Postsynaptic depolarizations are commonly referred to in their abbreviated form: _____.
13. Action potentials are generated in the axon initial segment, which is adjacent to the axon _____.
14. An action potential is elicited when the depolarization of the neuron reaches the _____.
15. Unlike postsynaptic potentials, which are graded, action potentials are _____ responses.
16. Neurons integrate postsynaptic potentials in two ways: through spatial summation and through _____ summation.

Scan Your Brain answers: (1) Parkinson's disease, (2) Parkinson's disease, (3) potential, (4) resting potential, (5) polarized, (6) motion, (7) outside, inside, (8) sodium, (9) ion channels, (10) sodium-potassium pumps, (11) hyperpolarize, (12) EPSPs, (13) hillock, (14) threshold of excitation, (15) all-or-none, (16) temporal.

4.4 Conduction of Action Potentials

How are action potentials produced, and how are they conducted along the axon? The answer to both questions is basically the same: through the action of **voltage-activated ion channels**—ion channels that open or close in response to changes in the level of the membrane potential (see Armstrong, 2007).

IONIC BASIS OF ACTION POTENTIALS

Recall that the membrane potential of a neuron at rest is relatively constant despite the high pressure acting to drive Na^+ ions into the cell. This is because the resting membrane is relatively impermeable to Na^+ ions and because those few that do pass in are pumped out. But things suddenly change when the membrane potential of the axon is depolarized to the threshold of excitation by an EPSP. The voltage-activated sodium channels in the axon membrane open wide, and Na^+ ions rush in, suddenly driving the membrane potential from about −70 to about +50 mV. The rapid change in the membrane potential associated with the *influx* of Na^+ ions then triggers the opening of voltage-activated potassium channels. At this point, K^+ ions near the membrane are driven out of the cell through these channels—first by their relatively high internal concentration and then, when the action potential is near its peak, by the positive internal charge. After about 1 millisecond, the sodium channels close. This marks the end of the *rising phase* of the action potential and the beginning of *repolarization* by the continued efflux of K^+ ions. Once repolarization has been achieved, the potassium channels gradually close. Because they close gradually, too many K^+ ions flow out of the neuron, and it is left hyperpolarized for a brief period of time. Figure 4.5 illustrates the timing of the opening and closing of the sodium and potassium channels during an action potential.

The number of ions that flow through the membrane during an action potential is extremely small in relation to the total number inside and around the neuron. The action potential involves only those ions right next to the membrane. Therefore, a single action potential has little effect on the relative concentrations of various ions inside and outside the neuron, and the resting ion concentrations next to the membrane are rapidly reestablished by the random movement of ions. The sodium–potassium pumps play only a minor role in the reestablishment of the resting potential.

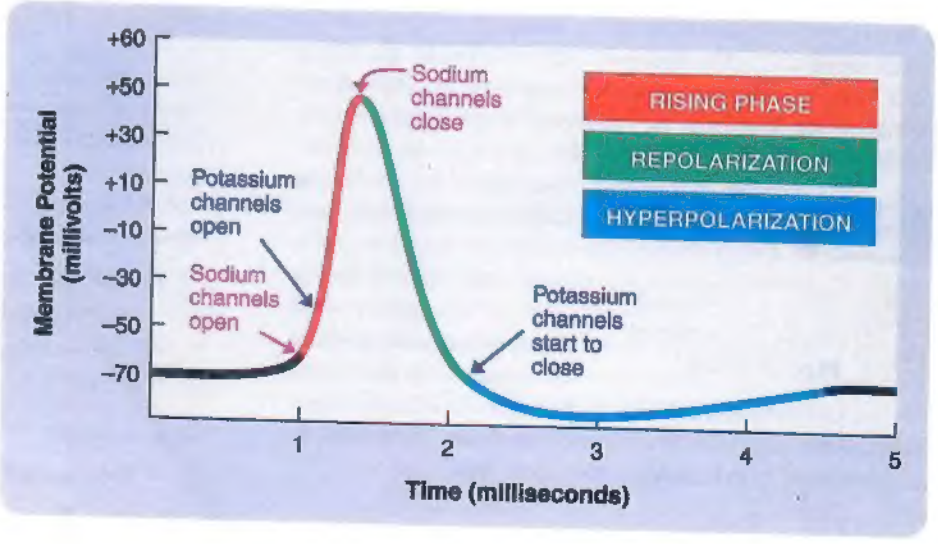

FIGURE 4.5 The opening and closing of voltage-activated sodium and potassium channels during the three phases of the action potential: rising phase, repolarization, and hyperpolarization.

REFRACTORY PERIODS

There is a brief period of about 1 to 2 milliseconds after the initiation of an action potential during which it is impossible to elicit a second one. This period is called the **absolute refractory period.** The absolute refractory period is followed by the **relative refractory period**—the period during which it is possible to fire the neuron again but only by applying higher-than-normal levels of stimulation. The end of the relative refractory period is the point at which the amount of stimulation necessary to fire a neuron returns to baseline.

The refractory period is responsible for two important characteristics of neural activity. First, it is responsible for the fact that action potentials normally travel along axons in only one direction. Because the portions of an axon over which an action potential has just traveled are left momentarily refractory, an action potential cannot reverse direction. Second, the refractory period is responsible for the fact that the rate of neural firing is related to the intensity of the stimulation. If a neuron is subjected to a high level of continual stimulation, it fires and then fires again as soon as its absolute refractory period is over—a maximum of about 1,000 times per second. However, if the level of stimulation is of an intensity just sufficient to fire the neuron when it is at rest, the neuron does not fire again until both the absolute and the relative refractory periods have run their course. Intermediate levels of stimulation produce intermediate rates of neural firing.

Simulate *Closer Look: Action Potential* at **MyPsychLab**

AXONAL CONDUCTION OF ACTION POTENTIALS

The conduction of action potentials along an axon differs from the conduction of EPSPs and IPSPs in two important ways. First, the conduction of action potentials along an axon is *nondecremental*; action potentials do not grow weaker as they travel along the axonal membrane. Second, action potentials are conducted more slowly than postsynaptic potentials.

The reason for these two differences is that the conduction of EPSPs and IPSPs is passive, whereas the axonal conduction of action potentials is largely active. Once an action potential has been generated, it travels passively along the axonal membrane to the adjacent voltage-activated sodium channels, which have yet to open. The arrival of the electrical signal opens these channels, thereby allowing Na^+ ions to rush into the neuron and generate a full-blown action potential on this portion of the membrane. This signal is then conducted passively to the next sodium channels, where another action potential is actively triggered. These events are repeated again and again until a full-blown action potential is triggered in all the terminal buttons (Huguenard, 2000). However, because there are so many ion channels on the axonal membrane and they are so close together, it is usual to think of axonal conduction as a single wave of excitation spreading actively at a constant speed along the axon, rather than as a series of discrete events.

The wave of excitation triggered by the generation of an action potential near the axon hillock always spreads passively back through the cell body and dendrites of the neuron. Although little is yet known about the functions of these backward action potentials, they are currently the subject of intensive investigation.

The following analogy may help you appreciate the major characteristics of axonal conduction. Consider a row of mouse traps on a wobbly shelf, all of them set and ready to be triggered. Each trap stores energy by holding back its striker against the pressure of the spring, in the same way that each sodium channel stores energy by holding back Na^+ ions, which are under pressure to move down their concentration and electrostatic gradients into the neuron. When the first trap in the row is triggered, the vibration is transmitted passively through the shelf, and the next trap is sprung—and so on down the line.

The nondecremental nature of action potential conduction is readily apparent from this analogy; the last trap on the shelf strikes with no less intensity than did the first. This analogy also illustrates the refractory period: A trap cannot respond again until it has been reset, just as a section of axon cannot fire again until it has been repolarized. Furthermore, the row of traps can transmit in either direction, just like an axon. If electrical stimulation of sufficient intensity is applied to the terminal end of an axon, an action potential will be generated and will travel along the axon back to the cell body; this is called **antidromic conduction.** Axonal conduction in the natural direction—from cell body to terminal buttons—is called **orthodromic conduction.** The elicitation of an action potential and the direction of orthodromic conduction are summarized in Figure 4.6.

CONDUCTION IN MYELINATED AXONS

In Chapter 3, you learned that the axons of many neurons are insulated from the extracellular fluid by segments of fatty tissue called *myelin*. In myelinated axons, ions can pass through the axonal membrane only at the **nodes of Ranvier**—the gaps between adjacent myelin segments. Indeed, in myelinated axons, axonal sodium channels are concentrated at the nodes of Ranvier (Salzer, 2002). How, then, are action potentials transmitted in myelinated axons?

When an action potential is generated in a myelinated axon, the signal is conducted passively—that is, instantly and decrementally—along the first segment of myelin to the next node of Ranvier. Although the signal is somewhat diminished by the time it reaches that node, it is

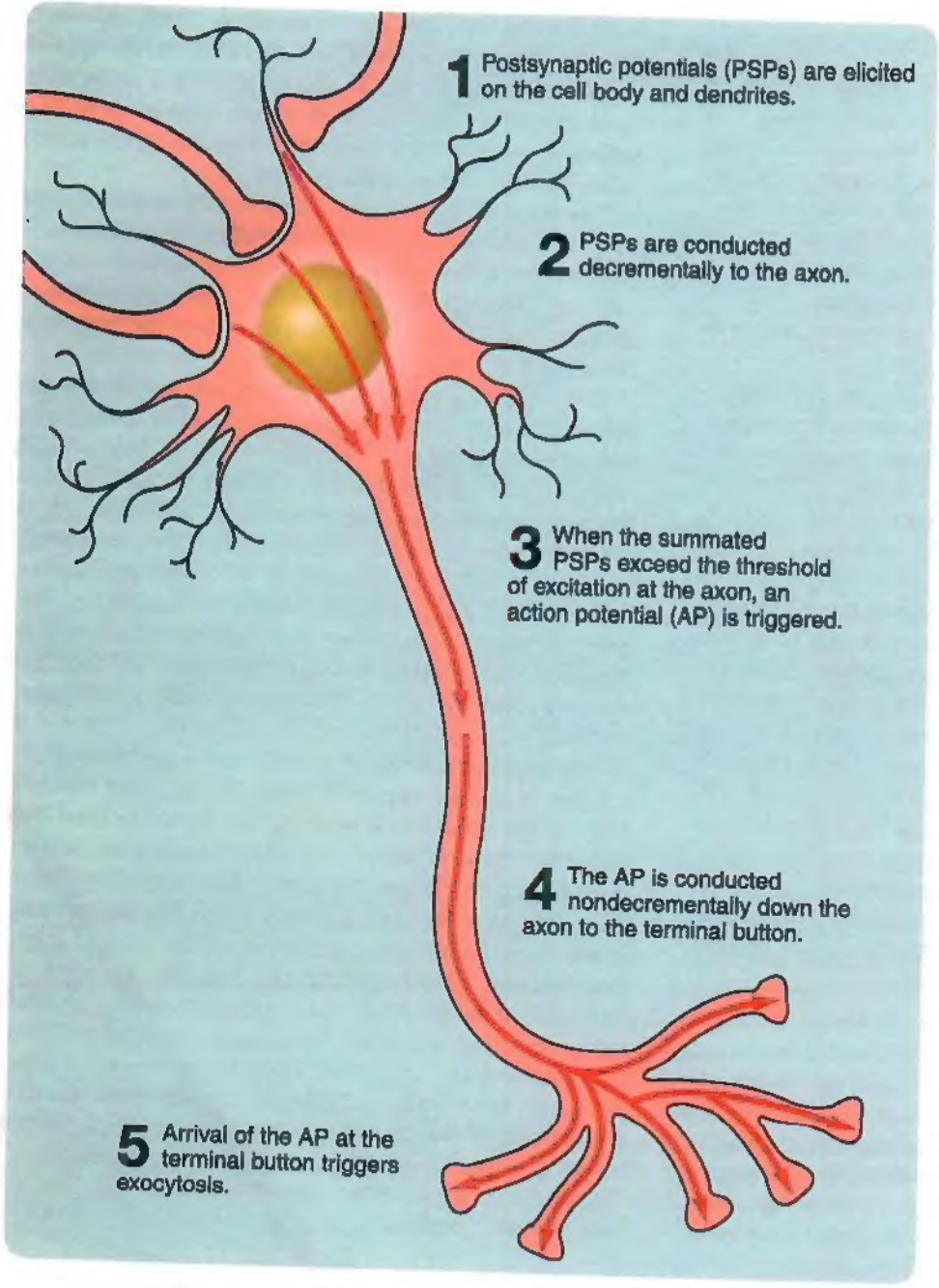

FIGURE 4.6 The direction of signals conducted orthodromically through a typical multipolar neuron.

still strong enough to open the voltage-activated sodium channels at the node and to generate another full-blown action potential. This action potential is then conducted passively along the axon to the next node, where another full-blown action potential is elicited, and so on.

Myelination increases the speed of axonal conduction. Because conduction along the myelinated segments of the axon is passive, it occurs instantly, and the signal thus "jumps" along the axon from node to node. There is, of course, a slight delay at each node of Ranvier while the action potential is actively generated, but conduction is still much faster in myelinated axons than in unmyelinated axons, in which passive conduction plays a less prominent role (see Poliak & Peles, 2003). The transmission of action potentials in myelinated axons is called **saltatory conduction** (*saltare* means "to skip or jump"). Given the important role of myelin in neural conduction, it is hardly surprising that diseases that damage the nervous system by attacking myelin have devastating effects on neural activity and behavior—see the discussion of multiple sclerosis in Chapter 10.

THE VELOCITY OF AXONAL CONDUCTION

At what speed are action potentials conducted along an axon? The answer to this question depends on two properties of the axon (see ffrench-Constant, Colognato, & Franklin, 2004). Conduction is faster in large-diameter axons, and—as you have just learned—it is faster in those that are myelinated. Mammalian *motor neurons* (neurons that synapse on skeletal muscles) are large and myelinated; thus, some can conduct at speeds of 100 meters per second (about 224 miles per hour). In contrast, small, unmyelinated axons conduct action potentials at about 1 meter per second.

There is a misconception about the velocity of motor neuron action potentials in humans. The maximum velocity of motor neuron action potentials was found to be about 100 meters per second in cats and was then assumed to be the same in humans. It is not. The maximum velocity of conduction in human motor neurons is about 60 meters per second (Peters & Brooke, 1998).

CONDUCTION IN NEURONS WITHOUT AXONS

Action potentials are the means by which axons conduct all-or-none signals nondecrementally over relatively long distances. Thus, to keep what you have just learned about

action potentials in perspective, it is important for you to remember that many neurons in mammalian brains either do not have axons or have very short ones, and many of these neurons do not normally display action potentials. Conduction in these *interneurons* is typically passive and decremental (Juusola et al., 1996).

THE HODGKIN-HUXLEY MODEL IN PERSPECTIVE

The preceding account of neural conduction is based heavily on the *Hodgkin-Huxley model*, the theory first proposed by Hodgkin and Huxley in the early 1950s (see Huxley, 2002). Perhaps you have previously encountered some of this information about neural conduction in introductory biology and psychology courses, where it is often presented as a factual account of neural conduction and its mechanisms, rather than as a theory. The Hodgkin-Huxley model was a major advance in our understanding of neural conduction (Armstrong, 2007). Fully deserving of the 1963 Nobel Prize, the model provided a simple effective introduction to what we now understand about the general ways in which neurons conduct signals. The problem is that the simple neurons and mechanisms of the Hodgkin-Huxley model are not representative of the variety, complexity, and plasticity of many of the neurons in the mammalian brain.

The Hodgkin-Huxley model was based on the study of squid motor neurons. Motor neurons are simple, large, and readily accessible in the PNS—squid motor neurons are particularly large. The simplicity, size, and accessibility of squid motor neurons contributed to the initial success of Hodgkin and Huxley's research, but these same properties make it difficult to apply the model directly to the mammalian brain. Hundreds of different kinds of neurons are found in the mammalian brain, and many of these have actions not found in motor neurons (see Nusser, 2009). Thus, the Hodgkin-Huxley model must be applied to cerebral neurons with caution. The following are some properties of cerebral neurons that are not shared by motor neurons:

Watch *Studying Squid* at **MyPsychLab**

- Many cerebral neurons fire continually even when they receive no input (Lisman, Raghavachari, & Tsien, 2007; Schultz, 2007).
- Axons of some cerebral neurons can actively conduct both graded signals and action potentials (Alle & Geiger, 2008).
- Action potentials of different classes of cerebral neurons vary greatly in duration, amplitude, and frequency (Bean, 2007).
- Many cerebral neurons do not display action potentials.
- The dendrites of some cerebral neurons can actively conduct action potentials (Urban & Castro, 2010).

Clearly, cerebral neurons are far more varied and complex than motor neurons, which have traditionally been the focus of neurophysiological research, and thus, results of studies of motor neurons should be applied to the brain with caution.

4.5 Synaptic Transmission: Chemical Transmission of Signals among Neurons

You have learned in this chapter how postsynaptic potentials are generated on the receptive membrane of a resting neuron, how these graded potentials are conducted passively to the axon, how the sum of these graded potentials can trigger action potentials, and how these all-or-none potentials are actively conducted down the axon to the terminal buttons. In the remaining sections of this chapter, you will learn how action potentials arriving at terminal buttons trigger the release of neurotransmitters into synapses and how neurotransmitters carry signals to other cells. This section provides an overview of five aspects of synaptic transmission: (1) the structure of synapses; (2) the synthesis, packaging, and transport of neurotransmitter molecules; (3) the release of neurotransmitter molecules; (4) the activation of receptors by neurotransmitter molecules; and (5) the reuptake, enzymatic degradation, and recycling of neurotransmitter molecules.

STRUCTURE OF SYNAPSES

Some communication among neurons occurs across synapses such as the one illustrated in Figure 4.7. At such synapses, neurotransmitter molecules are released from specialized sites on buttons into synaptic clefts, where they induce EPSPs or IPSPs in other neurons by binding to receptors on their postsynaptic membranes. The synapses featured in Figure 4.7 are *axodendritic synapses*—synapses of axon terminal buttons on dendrites. Notice that many axodendritic synapses terminate on **dendritic spines** (nodules of various shapes that are located on the surfaces of many dendrites)—see Figure 3.30. Also common are *axosomatic synapses*—synapses of axon terminal buttons on *somas* (cell bodies).

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Although axodendritic and axosomatic synapses are the most common synaptic arrangements, there are many others (see Matthews & Fuchs, 2010; Shepherd & Erulkar, 1997). For example, there are *dendrodendritic synapses*, which are interesting because they are often capable of transmission in either direction (see Urban & Castro, 2010). In addition, *axoaxonic synapses* are particularly important because they can mediate *presynaptic facilitation*